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Psychosocial distress and inflammation: Which way does causality flow?

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ABSTRACT

Objectives: This study queried causal direction in linkages of inflammation with psychosocial distress.
Methods: Data were from the 2005–2006 and 2010–2011 waves of the U.S. National Social Life, Health, and Aging Project. Inflammation was indicated by C-reactive protein, and distress by depression, anxiety, as well as stress. Autoregressive cross-lagged panel models were used to examine causal direction.
Results: Rather than being an outcome of psychosocial distress, inflammation was a predictor of it. Linkages were gender differentiated, with inflammation seeming to induce depression among men but stress among women.
Discussion: Contrary to previous literature, inflammation may not be a mechanism through which psychosocial distress gets "under the skin" to cause cardiovascular and metabolic issues. Rather, it may be a node through which social pathologies and life events influence both mental health and physiological problems.

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1. Introduction

A growing literature implicates low-grade inflammation, especially as indicated by C-reactive protein (CRP), as a major mechanism through which psychosocial distress gets "under the skin" to induce metabolic and cardiovascular issues (Kiecolt-Glaser et al., 2003; McDade, 2012; McDade et al., 2013). Inflammation has a demonstrated causative role in cardiovascular problems, as well as poor blood sugar control due to insulin resistance (Grundy et al., 2004; Yudkin, 2003). More specifically, recently developed high-sensitivity CRP assays suggest that chronic, low-grade inflammation is linked to subsequent incidence of cardiovascular disease (Danesh et al., 2000), type 2 diabetes (Pradhan et al., 2001), and the "metabolic syndrome" (McDade and Hayward, 2009; Ridker et al., 2003). However, distress also has a causal influence on these conditions, and excess distress-related mortality has been documented in multiple diseases (Cuijpers et al., 2014; Kiecolt-Glaser et al., 2003). Moreover, biological theory indicates the link between distress and inflammation is bidirectional—suggesting that distress may be a mediator rather than a precursor of inflammation-morbidity links (Berk et al., 2013; Kiecolt-Glaser et al., 2015; Miller et al., 2009; Raison et al., 2006; Raison and Miller, 2013; Shelton and Miller, 2010; Slavich and Irwin, 2014). Which pathway predominates at the population level remains unknown, since no nationally representative studies have examined causal direction. Without such evidence, inferences on the role of inflammation in stress-physiology linkages remain suspect.

The current study filled this gap by examining causal direction in linkages of CRP with psychosocial distress in late life. Specifically, it used data from the first two waves of the National Social Life, Health and Aging Project (NSHAP)—a national probability sample of older U.S. adults—along with autoregressive cross-lagged (ARCL) panel models (Curran, 2000) to simultaneously address reciprocal influences of CRP on distress and *vice versa*. The latter included standard indexes of depression, anxiety, as well as stress. Following recent work in causal modeling, between-wave participant attrition and corresponding selection issues were addressed through inverse-probability-of-attrition (IPA) weights.

2. Inflammation and distress: mechanisms

Biological theory indicates a direct psychobiological pathway from distress to inflammation. Norepinephrine has a demonstrated responsivity to one's stress ecology (Kiecolt-Glaser et al., 1997), and in turn activates gene expression of multiple inflammatory mediators through adrenergic stimulation (Kiecolt-Glaser et al., 2010). The process involves induction of inflammatory reactions as well as the simultaneous downregulation of antiinflammatory reactions, leading to increased cytokine release (Bierhaus et al., 2003). Animal

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as well as human lab studies also show that depression triggers inflammatory responses, leading to larger cytokine increases in reaction to stressors and pathogens. For instance, early adversity can enhance such responsiveness (Carpenter et al., 2010; Gouin et al., 2012a), and past or current mood disorders may function synergistically with stress to increase inflammation. In addition to direct psychobiological priming, distress can also influence inflammation through behavioral pathways—via diet and obesity (Yudkin, 2003), smoking and heavy alcohol-consumption (Chiu et al., 2008; Mukamal, 2006), lack of sleep (Meier-Ewert et al., 2004), and physical inactivity (Lancaster and Febbraio, 2014).

Inversely, elevated inflammation (as indexed by both proinflammatory cytokines and CRP) predicts incident depression (Valkanova et al., 2013). Cytokines such as IL-6 induce depressive symptoms through a range of affect-related processes. Enhanced inflammatory signaling dysregulates neurotransmitter metabolism, impairs neuronal health, and alters neural activity in affect-relevant brain regions (Miller et al., 2013; Slavich and Irwin, 2014). For instance, cytokines alter production, metabolism, and transport of neurotransmitters that synergistically affect mood—including dopamine, glutamate, and serotonin (Capuron and Miller, 2011). Inflammation also influences neuronal growth and survival. Cytokines contribute to oxidative stress, which in turn damages glial cells in affect-relevant brain regions, such as the prefrontal cortex and the amygdala (Leonard and Maes, 2012). In addition to their influence on neural processes, cytokines promote dysregulated hypothalamic-pituitary-adrenal (HPA) axis functioning, a key feature of depression (Pace et al., 2007; Stetler and Miller, 2011). Importantly, inflammation is neither necessary nor sufficient to induce or prolong depression (Glassman and Miller, 2007; Raison and Miller, 2011). However, it demonstrably plays an important role in a substantial subpopulation (Haroon et al., 2012). Potential moderators include individual physiology: some people have bodily systems that protect them from developing inflammation-based depression, while others do not. Even lower levels of inflammation could be depressogenic in vulnerable individuals, a phenomenon that has been termed “immune response element amplification” (Raison and Miller, 2011).

3. Inflammation and distress: evidence

Only a few longitudinal studies examine causal direction in distress-inflammation associations. All are based on community or nonprobability samples, and findings contradict each other. This is not surprising if this linkage is indeed moderated by unexamined physiological factors—or by life course experiences such as early adversity, which have known variations across sociodemographic groups (Hayward and Gorman, 2004; Willson et al.,2007). Causal direction on a population-wide basis remains unclear.

First, Copeland et al. (2012) used nine waves of data from the Great Smoky Mountains Study (N = 1420) to examine CRP's responsiveness to current depression as well as cumulative episodes of depression. The sample was restricted to children in the community aged 9 to 16, 19, and 21 years old, recruited from 11 counties in western North Carolina. CRP levels were not associated with later depression status. In contrast, all depression indicators had associations with later CRP levels. Linkages with current depression were attenuated after controlling body mass index, smoking, and medication use. The effect of cumulative depressive episodes, however, remained significant after accounting for a range of covariates.

Second, in data from the Whitehall II cohort of over 3000 British white-collar civil servants, baseline CRP predicted cognitive symptoms of depression at 12-year follow-up after controlling baseline depression (Gimeno et al., 2009). Cognitive symptoms of depression, however, did not predict CRP levels at follow-up, suggesting a unidirectional effect from inflammation to psychiatric status.

Third, Matthews et al. (2010) used nonrepresentative data on 1781 older women from the multi-ethnic Study of Women's Health Across the Nation (SWAN) to examine causal direction in depression-CRP linkages. Participants were from seven study sites. At each, recruits included White women as well as those from one ethnic minority group—Black women in Boston, MA, Chicago, IL, Pittsburgh, PA, and Detroit area, MI; Chinese in the Oakland/East Bay region, CA; Hispanic women in Newark, NJ, and Japanese in Los Angeles, CA. Analyses were restricted to initially healthy women. The authors found support for both causal directions over a 1-year to 2-year follow-up period. However, the association of depressive levels with later CRP was only marginally significant.

Fourth, data from 2544 healthy Black and White participants in the nonprobability Coronary Artery Risk Development in Young Adults (CARDIA) study were used to examine the prospective association of depressive symptoms with circulating CRP five years later (Janicki Deverts et al., 2010). Participants were between 18 and 30 years at baseline and were recruited from four centers: Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA. Depression predicted later CRP levels at follow up, but only among Blacks. There was no evidence that CRP predicted later depression in either Black or White participants.

Additional longitudinal evidence comes from two studies based on clinical samples with strong potential for selection bias. In data from 667 outpatients with established coronary heart disease from the Heart and Soul Study, more baseline depressive symptoms predicted higher inflammation (IL-6 and CRP) at follow up, but not *vice versa* (Duivis et al., 2011). Similarly, in a sample of 163 adults with acute coronary syndrome, Shaffer et al. (2011) found that total depressive symptom severity at the time of a cardiac event predicted a smaller *decrease* in CRP 1 month later. There was no evidence that CRP influenced change in depressive symptoms.

In addition to these bidirectional analyses, a few longitudinal studies have examined unidirectional associations between distress and inflammation. In data from 236 adult participants in the Detroit Neighborhood Health Study, inflammation (IL-6 and CRP) levels at baseline were unassociated with incidence of depression (Simanek et al., 2014). Results from the Sydney Memory and Aging Study, comprised of non-demented community dwelling older adults from the eastern suburbs of Sydney, Australia (N = 1037), suggested that the inflammatory marker IL-8 may predict first onset of depressive symptoms (Baune et al., 2012). Finally, a prospective population-based study of older adults in Tuscany, Italy (N = 991) found that those with high plasma levels of the inflammatory marker IL1-ra may have a higher risk of developing depressive symptoms over time (Milaneschi et al., 2009).

To address these contradictions and gaps in the literature, this study tested two hypotheses:

Hypothesis 1. *More distress at baseline, as indicated by higher scores on scales of depression, anxiety and stress, will predict higher inflammation (CRP) at follow up.*

Hypothesis 2. *Higher inflammation at baseline will predict more distress at follow up.*

4. Methods

4.1. Data

Data were from the first two waves of the U.S. National Social Life, Health, and Aging Project (NSHAP), fielded in 2005–2006 and

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